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EXAMINER	
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ART UNIT	PAPER NUMBER
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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/506,962	ULLRICH ET AL.	
	Examiner	Art Unit	
	LYNN BRISTOL	1643	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *11/17/08 and 12/11/08* .

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *2,8 and 10-17* is/are pending in the application.

4a) Of the above claim(s) *11 and 12* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *2,8,10 and 13-17* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *11/17/08* .
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

Continued Examination Under 37 CFR 1.114

1. A request for continued examination under 37 CFR 1.114, including the fee set forth in 37 CFR 1.17(e), was filed in this application after final rejection. Since this application is eligible for continued examination under 37 CFR 1.114, and the fee set forth in 37 CFR 1.17(e) has been timely paid, the finality of the previous Office action has been withdrawn pursuant to 37 CFR 1.114. Applicant's submission filed on 11/17/08 and 12/11/08 has been entered.

2. Claims 2, 8, and 10-17 are all the pending claims in this application.

3. The response of 12/11/08 to the Notice of Non-Compliant Amendment is acknowledged.

4. Claims 2, 8, 13, 15 and 17 were amended in the Response of 11/17/08 and 12/11/08 and the status identifier for amended Claim 15 was corrected in the Response of 12/11/08.

5. Claims 11 and 12 are withdrawn from examination.

6. Claims 2, 8, 10 and 13-17 are all the pending claims under examination.

7. This Office Action contains new grounds for objection.

Information Disclosure Statement

8. The IDS of 11/17/08 has been considered and entered. The initialed and signed 1449 form is attached.

Withdrawal of Objections

Claim Objections

9. The objection to Claims 13 and 17 because the claims recite an apparent typographical error for "a subject*ed* in need thereof" is withdrawn. The claims have been amended to recite "a subject in need thereof."

Withdrawal of Rejections

Claim Rejections - 35 USC § 112, second paragraph

10. The rejection of Claims 13 and 15 in lacking antecedent basis for the limitation "the growth factor receptor" is withdrawn. The rejection of Claim 13 was inadvertent and the rejection of Claim 15 is moot in view of the amendment of the claim to depend from Claim 14.

11. The rejection of Claim 15 in lacking antecedent basis for the limitation "the EGFR family" is withdrawn in view of the amendment of the claim to depend from Claim 14.

Rejections Maintained

Claim Rejections - 35 USC § 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Written Description

12. The rejection of Claims 2, 8, 10 and 13-17 under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement (i.e., Claims 2, 8, 10 and 13-17 recite subject matter for "an antibody which binds pro-HB-EGF and which blocks the processing of said pro-HG-EGF" that is not defined in the specification (*In re Morris* 127 F.3d 1048, 44USPQ2d 1023 (Fed. Cir. 1997) and MPEP 2163)) is maintained.

For purposes of review, the rejection was set forth in the Office Action of 7/16/08 as follows:

"Claims 2, 8, 10 and 13-17 recite subject matter for "an antibody which binds pro-HB-EGF and which blocks the processing of said pro-HG-EGF" that is not defined in the specification (*In re Morris* 127 F.3d 1048, 44USPQ2d 1023 (Fed. Cir. 1997) and MPEP 2163).

The specification discloses "an antibody which binds pro-HB-EGF and which blocks the processing of said pro-HG-EGF." The specification does not otherwise cite a commercial example(s) of such an antibody or reduce to practice any antibody meeting all of these properties. The prior art does not support the existence of any such antibody.

Under the Written Description Guidelines (66 FR 1099 (Jan. 5, 2001); 1242 O.G. 168 (Jan. 30, 2001)), the claimed invention must meet the following criteria as set forth.

Actual reduction to practice: the specification does not show any embodiments that meet the limitations for "an antibody which binds pro-HB-EGF and which blocks the processing of said pro-HG-EGF" reduced to practice.

Disclosure of drawings or structural chemical formulas: the specification and drawings do not show that applicant was in possession of the claimed invention as a whole (i.e., using the antibody to prevent or treat cancer).

Sufficient relevant identifying characteristics: the specification does not identify 1) a complete structure, ii) partial structure, iii) physical and/or chemical properties, or iv) functional characteristics coupled with correlation between structure and function for the antibody.

Method of making the claimed invention: the specification does not teach or suggest how to make "an antibody which binds pro-HB-EGF and which blocks the processing of said pro-HG-EGF".

Level of skill and knowledge in the art: the examiner's search of commercial literature databases (Medline, CAPLUS), the ATCC website and the ExactAntigen database did not reveal the existence of any "antibody which binds pro-HB-EGF and which blocks the processing of said pro-HG-EGF."

Predictability in the Art: one of skill in the art could reasonably expect to generate an antibody that binds the pro-HB-EGF protein but it is not predictable that the antibody would also inhibit processing of said pro-HG-EGF or that the same antibody could be administered in a subject and still exhibit the same properties.

Applicants' specification does not show the existence of a commercial antibody which binds pro-HB-EGF and which blocks the processing of said pro-HG-EGF. Applicants' specification has not reduced to actual practice a working example of an antibody with these characteristics. One of skill in the art could reasonably conclude that Applicants were not in possession of "an antibody which binds pro-HB-EGF and which blocks the processing of said pro-HG-EGF" at the time of application filing.

Applicants allegations on pp. 4-7 of the Response of 11/17/08 that the identity of the antigen is well known (e.g., the interpretations of decisions of Noelle v. Lederman; In

re Bucher; Hybritech, Inc. v Monoclonal Antibodiesd, Inc.; and Capon v. Eshar; the PUBMED search output for pro-HB-EGF (Exhibit A); the GenBank accession no. for pro-HB-EGF (M60278) in WO 01/35889; methods for making antibodies (Kohler et al., (Nature, vol. 256; pp. 495-497, 1975) and Mathews and Wells (Science, vol. 260, pages: 1113-1117, 1993)); processing of nerve growth factor (Exhibit B (PUBMED search) and the review article by Seidah et al. (Biochem J., 1996)), is not considered to support an antibody which meets all of the functional limitations required of the instant method claims.

Response to Arguments

Applicants have failed to show the existence of appropriate epitopes/regions of the target entigen, pro-HB-EGF, that would provide the claimed functional properties required of the antibody. The Court has held that the disclosure of screening assays and general classes of compounds was not adequate to describe compounds having the desired activity: without disclosure of which peptides, polynucleotides, or small organic molecules have the desired characteristic, the claims failed to meet the description requirement of § 112. See University of Rochester v. G.D. Searle & Co., Inc., 69 USPQ2d 1886,1895 (Fed. Cir. 2004). The problem here is that the instant specification fails to provide a disclosure of which antibody retain the appropriate antibody specificity for pro-HB-EGF *and* which blocks the processing of pro-HB-EGF. A skilled artisan cannot, as one can do with a fully described genus, visualize or recognize the identity of the members of the genus that exhibit this functional property. There is insufficient guidance and direction as to the written description of the claimed antibody,

as broadly encompassed by the claimed invention. Given the well known high level of polymorphism of immunoglobulins / antibodies, the skilled artisan would not have been in possession of the vast repertoire of antibodies and the unlimited number of antibodies encompassed by the claimed invention; one of skill in the art would conclude that applicant was not in possession of the functional attributes of a representative number of species possessed by the members of the genera of "an antibody which binds to pro-HB-EGF and which blocks the processing of said pro-HB-EGF" as indicated above, and broadly encompassed by the claimed invention. One of skill in the art would conclude that the specification fails to disclose a representative number of species much less a single species to describe the claimed genera.

Enablement

13. The rejection of Claims 2, 8, 10 and 13-17 under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement is maintained.

The rejection was set forth in the Office Action of 7/16/08 as follows:

"Nature of the Invention/ Skill in the Art

Claims 13-16 and (dependent claims 2, 8 and 10) are interpreted as being drawn to a method for the prevention or treatment of cell proliferation, cell migration, invasivity or anti-apoptosis in any cancer, where the cancer is associated with increased G-protein mediated signal transduction and is colon, kidney, bladder, prostatic, breast, lung, or ovarian cancer and where the subject is administered a composition comprising an antibody which binds pro-HB-EGF and which blocks the processing of said pro-HG-EGF (Claim 13), where the antibody inhibits activation of a GFR of the EGFR family (Claim 14), and the GFR is HER-2, HER-3 or HER-4 (claim 15), where the method is for the treatment (Claim 16), where the GFR is EGFR (Claim 2), where the composition is a pharmaceutical composition comprising the antibody (Claim 8) and the cancer is a human cancer (Claim 10).

Claim 17 is interpreted as being drawn to a method for the treatment of cell proliferation, cell migration, invasivity or anti-apoptosis in any cancer, where the cancer is associated with increased G-protein mediated signal transduction and is colon, kidney, bladder, prostatic, breast, lung, or ovarian cancer and where the subject is administered a composition comprising an antibody which binds pro-HB-EGF and which blocks the processing of said pro-HG-EGF.

The relative skill in the art is a clinical oncologist.

Disclosure in the Specification

The specification contemplates using an antibody capable of binding to pro-HB-EGF and which inhibits processing of precursor as an embodiment for affecting a growth-factor receptor ligand precursor. The specification contemplates using the antibody in order to treat or prevent a disorder associated with G-mediated signal

transduction effecting EGFR where the agent effects a process of cell proliferation, cell migration, invasivity and/or anti-apoptosis. No where in the specification are any methods using an in vitro cell-based assay much less an animal model correlate for any disorder encompassed by the claims showing that the antibody could be practiced in the claimed method and that a prophylactic or therapeutic effect would be accomplished. One of skill in the art could not practice the invention because Applicants have not identified an example of an antibody having the instant claimed properties much less where the antibody is administered to any subject having any cancer. Applicants' prophetic antibody has the alleged properties of preventing just any cancer and treating just any cancer where the general field of art recognizes the unpredictability of preventing/treating just any cancer much less using an immunotherapeutic/ immunoprophylactic antibody in a human subject.

Prior Art Status: Cancer Treatment and Prevention is Unpredictable

A tumor is a 3-dimensional complex consisting of interacting malignant and non-malignant cells. Vascularization, perfusion and drug access to the tumor cells are not evenly distributed and this is an important source of heterogeneity in tumor response to drugs. Therefore, the antibody effect(s) in any cancer subject much less a human in the absence of any in vitro cell-based testing or in vivo animal cancer model correlates as in the present case, is not reliable or predictable and further evaluation in cell assays systems and animal tumor systems is essential.

Further, it is not clear what the best approaches are to examining a drug or antibody effect in preclinical testing. Voskoglou-Nomikos (Clin. Can. Res. 9:4227-4239 (2003); cited in the PTO 892 form of 1/9/08) conducted a study using the Medline and Cancerlit databases as source material in comparing the clinical predictive value of three pre-clinical laboratory cancer models: the in vitro human cell line (Figure 1); the mouse allograft model; and the human xenograft model (Figures 2 and 3). Significantly when each of the cancer models was analyzed against Phase II activity, there was a negative correlation for the in vitro human cell line models being predictive of good clinical value. No significant correlations between preclinical and clinical activity were observed for any of the relationships examined for the murine allograft model. And the human xenograft model showed good tumor-specific predictive value for NSCLC and ovarian cancers when panels of xenografts were used, but failed to predict clinical performance for breast and colon cancers. Voskoglou-Nomikos suggests that "the existing cancer models and parameters of activity in both the preclinical and clinical settings may have to be redesigned to fit the mode of action of novel cytostatic, antimetastatic, antiangiogenisis or immune-response modulating agents" and "New endpoints of preclinical activity are contemplated such as the demonstration that a new molecule truly hits the intended molecular target" (p.4237, Col. 1, ¶6).

Dennis (Nature 442:739-741 (2006); cited in the PTO 892 form of 1/9/08) also recognizes that human cancer xenograft mouse models for testing new drugs has been and will remain the industry standard or model of choice, but it is not without problems because "many more [drugs] that show positive results in mice have little or no effect in humans". (p. 740, Col. 1, ¶3). Dennis describes transgenic animal mouse models as an alternative to xenograft modeling and the general differences between mice and humans when it comes to tumor modeling: 1) cancers tend to form in different types of tissue, 2) tumors have fewer chromosomal abnormalities, 3) ends of chromosomes (telomeres) are longer, 4) telomere repairing enzyme active in cells, 5) short lifespan, 6) fewer cell divisions (10^{11}) during life than humans (10^{16}), 7) metabolic rate seven time higher than humans, and 8) lab mice are highly inbred and genetically similar.

Cespedes et al. (Clin. Transl. Oncol. 8(5):318-329 (2006)) review the some of the examples of art-recognized animal disease model correlates for the corresponding human disease in Tables 1-3. Cespedes emphasizes the challenges in using animal models as predictive correlates for human responsiveness to therapeutics and sets forth on pp. 318-319 a list of criteria that would represent the ideal in vivo model for studying cancer therapeutics. As regards the use of xenograft modeling, Cespedes teaches:

> "One limitation of the xenograft models is precisely their use of an immunocompromised host, which eliminates the possibility of studying the role of the immune system in tumor progression. Some authors also think that cancer and host cells being from different species may limit the occurrence of critical tumor-stroma interactions, leading to an inefficient signaling. The organ of implantation could also become a limitation to the system. Thus, as it has already been described, subcutaneous xenografts infrequently metastasize and are unable to predict response to drugs" (p. 325, Col. 1, ¶2).

One skilled in the art would reasonably conclude that evidence obtained in in vitro cell-based assays or even mouse cancer models using the prophetic antibody of applicants invention would not even necessarily correlate with results expected in any human tumor.

Skill in the Art/Undue Experimentation

It appears that undue and inordinate experimentation would be required of one skilled in the art to practice the instant invention using the teachings of the specification alone and the specification fails to enable the use of the method for any tumor therapy and any tumor prevention much less in any human. Due the unpredictability of cancer therapeutics in general, as evidenced by Voskoglou-Nomikos, Dennis and Cespedes, *and* in view of the absence of

guidance for procuring or making an antibody which binds pro-HB-EGF and which blocks the processing of said pro-HG-EGF and the absence of working examples concerning the use the prophetic antibody in the method invention, one skilled in the art would not know how to practice the broadly claimed invention. One skilled in the art could not administer to any subject having any cancer "an antibody which binds pro-HB-EGF and which blocks the processing of said pro-HG-EGF" for the treatment and/or prevention of any cancer much less a human cancer and its accompanying pathologies, without undue experimentation."

Applicants allegations on pp. 7-9 of the Response of 11/17/08 the attached reference article (Molina et al. (Cancer Research, 61:4744-4749 (2001)) and the HERCEPTIN® product data sheet have been carefully considered and are not found persuasive.

A) Applicants have argued that under In re Marzocchi, In re Brana, and In re Bundy that they are not required to produce working examples showing the use of the antibody to treat a cancer and that the Office has not met the burden in showing lack of enablement.

Response to Arguments

The invention is in a class of invention which the CAFC has characterized as "the unpredictable arts such as chemistry and biology." Mycogen Plant Sci., Inc. v. Monsanto Co., 243 F.3d 1316, 1330 (Fed. Cir. 2001). Absent a showing to the contrary, arguments of counsel alone are not found to be sufficient in overcoming the enablement rejection (MPEP 2144.03).

Applicants have not addressed how to practice using much less the existence of an antibody meeting all of the claim limitations for an antibody that i) binds pro-HB-EGF; ii) blocks the processing of pro-HB-EGF, iii) directly or indirectly effects G-protein mediated signal transduction in a colon cancer, kidney cancer, bladder cancer, prostate cancer, breast cancer, lung cancer or ovarian cancer, iv) directly or indirectly affects cell

proliferation, cell migration, invasivity or anti-apoptosis in a cancer, and v) inhibits activation of a growth-factor receptor of the EGFR family such as HER-2 or EGFR where the method endpoint is treatment of cancer.

Applicants have not addressed how the treatment of any recited cancer with the antibody therapy is enabled when the references of record (Voskoglou-Nomikos; Dennis and Cespedes) teach the unpredictablity of drug therapy much less the complexity in choosing animal model correlates to human diseases. Applicants have not addressed how these references are irrelevant to practicing the method invention in any subject as instantly encompassed by the claims, because Applicants have ignored the general teaching of unpredictability for this art.

Applicants have not addressed how the inventive antibody is better or improved in its ability to access a heterogeneous population of tumor cells in vivo, to internalize within the tumor cell and to block the processing of the pro-HB-EGF protein into a mature HB-EGF protein in order to affect a direct or indirect therapeutic response.

B) Applicants allege "Molina (Cancer Research, 2001) describes the ability of a therapeutically effective antibody Trastuzumab, which binds selectively with high affinity to the extracellular domain of Her2, to inhibit the cleavage of the extracellular domain of the receptor tyrosine kinase Her2 in vitro. The therapeutic antibody has now been utilized for the treatment of tumor patients. See, the disclosure contained Table 1 and Table 2 of the enclosed product brochure on Trastuzumab."

Response to Arguments

The reference article and product data sheet demonstrate a single example of an art recognized antibody, Trastuzumab, that explicitly or inherently meets the limitations in the method for Claims 14 and 15 where the antibody inhibits activation of a growth factor receptor of the EGFR family and the growth factor receptor is HER-2. Molina teaches Trastuzumab was able to effectively block basal and induced HER2 cleavage, and this property was not shared by 2CA, another antibody against the HER2 ectodomain; Trastuzumab is effective in the therapy of breast tumors that overexpressing HER2. The Genetech Trastuzumab product datasheet teaches clinical trial indication for breast cancer and metastatic breast cancer in human patients. Notably, Applicants specification does not even contemplate or provide literal support for Trastuzumab much less any other art recognized antibodies targeting HER-2.

However, the instant method claims also require that *the very same antibody*, and in this example, Trastuzumab, would also have the property of i) binds pro-HB-EGF; ii) blocks the processing of pro-HB-EGF, iii) directly or indirectly effects G-protein mediated signal transduction in a colon cancer, kidney cancer, bladder cancer, prostate cancer, breast cancer, lung cancer or ovarian cancer, iv) directly or indirectly affects cell proliferation, cell migration, invasivity or anti-apoptosis in a cancer, and v) achieves the therapeutic endpoint of the method. Applicants have not shown that the Trastuzumab would also block the processing of pro-HB-EGF. The examiner does not believe that pro-HB-EGF (heparin binding-EGF-like growth factor) and HER-2 (C-cerb-2 or Tyrosine kinase-type cell surface receptor HER2) is the same protein molecule, and therefore, it is not understood how the Trastuzumab antibody could affect the two different protein

substrates as implied by Applicants. The specification and the cited art does not enable the use of Trastuzumab to bind pro-HB-EGF or block the processing of pro-HB-EGF absent a showing to the contrary.

Claims 14 and 15 would be considered enabled for the aspect of treating breast cancer in vivo using Trastuzumab but the generic Claims 13 and 17 require the same antibody to meet numerous other functional criteria that do not appear to be properties of the Trastuzumab.

The scope of the claims with respect to the kinds of antibodies and the lack of evidence showing the genus of antibodies meeting these requirements does not enable the full scope of the method as discussed above. MPEP 2164.04 states in part: "The scope of the required enablement varies inversely with the degree of predictability involved, but even in unpredictable arts, a disclosure of every operable species is not required. A single embodiment may provide broad enablement in cases involving predictable factors, such as mechanical or electrical elements. In re Vickers, 141 F.2d 522, 526-27, 61 USPQ 122, 127 (CCPA 1944); In re Cook, 439 F.2d 730, 734, 169 USPQ 298, 301 (CCPA 1971). However, in applications directed to inventions in arts where the results are unpredictable, the disclosure of a single species usually does not provide an adequate basis to support generic claims. In re Soll, 97 F.2d 623, 624, 38 USPQ 189, 191 (CCPA 1938). In cases involving unpredictable factors, such as most chemical reactions and physiological activity, more may be required. In re Fisher, 427 F.2d 833, 839, 166 USPQ 18, 24 (CCPA 1970) (contrasting mechanical and electrical elements with chemical reactions and physiological activity). See also In re Wright, 999

F.2d 1557, 1562, 27 USPQ2d 1510, 1513 (Fed. Cir. 1993); In re Vaeck, 947 F.2d 488, 496, 20 USPQ2d 1438, 1445 (Fed. Cir. 1991). This is because it is not obvious from the disclosure of one species, what other species will work."

New Grounds for Objection

Claim Objections

14. Claims 13 and 16 are objected to under 37 CFR 1.75(c), as being of improper dependent form for failing to further limit the subject matter of a previous claim. Applicant is required to cancel the claim(s), or amend the claim(s) to place the claim(s) in proper dependent form, or rewrite the claim(s) in independent form. Claims 13 and 16 are both drawn to a method for treatment of cell proliferation, cell migration, invasivity or anti-apoptosis in a cancer.

Conclusion

15. No claims are allowed.

16. The closest art is considered by the Examiner to be:

Lackmann et al. (US 20080317763; published December 25, 2008; filed December 19, 2005) who teach an antibody binding the substrate recognition site of the ADAM protease, which binding reduces affinity for the substrate and prevents substrate cleavage and where the substrate is pro-HB-EGF and where the processing of pro-HB-EGF is found in cancers.

17. Any inquiry concerning this communication or earlier communications from the examiner should be directed to Lynn Bristol whose telephone number is 571-272-6883. The examiner can normally be reached on 8:00-4:00, Monday through Friday.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Larry Helms can be reached on 571-272-0832. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

/Lynn A. Bristol/
Examiner, Art Unit 1643
Partial Signatory Authority